Gaze Paresis in Amitriptyline Overdose

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An important clinical tenet emphasized by Plum and Posner [2] is that oculomotor dysfunction in the unconscious patient indicates a structural rather than a metabolic cause of coma, unless the latter is profound. Of the few agents [2] that may produce absent oculovestibular responses in metabolic coma, amitriptyline is included based on a single case report [1]. We support this observation by providing another case.

A 26-year-old woman with chronic depression ingested an unknown quantity of amitriptyline. Her vital signs were normal. She responded incoherently to verbal commands and moved all extremities purposefully to pain. The pupils were 5 mm bilaterally and were sluggishly reactive to light. Oculocephalic and oculovestibular maneuvers revealed no horizontal or vertical eye movements. Corneal reflexes were present, as were symmetrical facial movements. Myotatic reflexes were symmetrically hyperactive with flexor plantar responses. A urinary drug screen was positive for amitriptyline. After 24 hours she became coherent and her gaze paresis resolved.

The mechanism for gaze paresis from amitriptyline overdose is uncertain. Experimental evidence has shown depression of midbrain reticular unit activity in cats poisoned with the drug [4]. The pontine reticular formation may also be impaired by amitriptyline; thus, physiological interruption in these two areas of the brainstem may involve the horizontal and vertical gaze centers. Amitriptyline may also act as a direct suppressor of vestibular nuclei [3].

The point made by Mladinich and Carlow [1] deserves reemphasizing: "total paresis of eye movements in light coma should serve as an important sign of amitriptyline poisoning in a psychiatric patient." Barbiturates, phenytoin, and succinylcholine [2] should also be considered in similar cases of light coma with gaze paresis.

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Editor's Note

Plum and Posner agree that transient external ophthalmoplegia occurs fairly frequently in amitriptyline intoxication, even in patients who are no more than obtunded. Several patients have been observed at the New York Hospital in whom drug analyses indicated no evidence of co-contamination by barbiturates, phenytoin, or other identifiable sedatives.

Variability of Charcot-Marie-Tooth Disease

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Electrophysiological and pathological evidence—and, to a lesser extent, clinical features—indicate that peroneal muscle atrophy, or Charcot-Marie-Tooth disease, is a heterogeneous syndrome in which several different disease groups can be recognized [1–4]. This makes it essential that reports such as Williams' [5] of abnormalities in "Charcot-Marie-Tooth disease" separate patients into the previously recognized subgroups. I hope Dr Williams will present his data separated in this fashion.

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Reply

Lowell L. Williams, MD

I agree with Dr Bradley's first statement and his cited references. The Charcot-Marie-Tooth disease probands of my study, although satisfying the characteristics described by Drs P. J. Dyck and E. H. Lambert (Dr Bradley's references 2 and 3) of hypertrophic neuropathy types I and II, did vary considerably in the details of their physiological and clinical expression. It was because of this variability that an HLA consensus was sought. But, despite careful analysis of the results, attempts to connect individual HLA loci with specific symptoms, nerve conduction velocities, or Charcot-Marie-Tooth disease subgroups were not successful. Therefore, the heterogeneity of the syndrome remains an enigma.

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